Diagnosis and Prevention of Avian Leukosis

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Abstract This paper introduced the characteristics of avian leukosis from the aspects of pathogen, epidemiology, clinical symptoms, and anatomical symptoms, and puts forward clinical comprehensive diagnosis and laboratory diagnostic methods. Besides, the disease is also differentiated from similar diseases of chicken such as Marek's disease, infectious bursal disease and reticuloendothelial hyperplasia. Finally, the prevention and control measures against the disease were proposed.

Keywords Avian leukosis; Pathogen; Epidemiology; Clinical symptom; Anatomical symptom; Diagnosis method; Prevention and control measure

1 Pathogen

1.1 Virus structure Avian leukosis is a disease caused by avian leukosis virus belonging to the family of Retroviridae. The virus particle is nearly spherical, 80–100 nm in diameter, consisting of an external envelope and an internal electron-dense core. The core is 45 nm in diameter and is located in the center of the virus particle. There are radial projections on the outside of the viral envelope, which are about 8 nm in diameter, and the virus is released from the envelope in a budding manner.

1.2 Virus clustering The virus can be classified into 10 subgroups from A to J based on the form of neutralization reaction, host range, envelope characteristics, and other criteria, of which 6 subgroups, A, B, C, D, E, and J, are found in chickens. Subgroups A and B are common exogenous viruses that mainly infect light commercial laying hens and cause lymphatic leukemia in chickens; subgroups C and D are rarely reported exogenous viruses; subgroup E consists of prevalent oncogenic endogenous viruses; and subgroup J avian leukosis virus is the dominant subgroup in chicken flocks in China.

1.3 Physicochemical properties The buoyant density of avian leukosis virus in sucrose is 1.15–1.17 g/cm^3. The virus is sensitive to heat, fat-soluble solvents, detergents, and formaldehyde. Proteolytic enzymes are able to remove some of the glycoprotein from the surface of virus particles, which are fairly resistant to ultraviolet light. Most cases of avian leukosis virus infection of cells do not cause damage to the cells but will become a recessive infection. However, in some cases or with certain types of avian leukosis virus infection, it can lead to cell transformation or cause cellular lesions, which in turn can lead to immunosuppression.

2 Epidemiology

2.1 Source of infection and transmission routes Diseased and virulent chickens are the main source of infection of the disease. Eggs and chicks from hens with viremia are often virulent. Such congenitally infected chicks are often immune-tolerant, do not produce anti-tumor virus antibodies, and can also be important sources of infection. Avian leukosis virus has two modes of transmission, vertical transmission and horizontal transmission. Vertical transmission is the main mode of transmission, in which the virus is passed vertically from parent to offspring, and the infected hen passes the virus to the embryo through the eggs. The hatched chick carries the virus for the rest of its life and passes it on from generation to generation. Horizontal transmission is slower, and this method ensures that transmission is maintained, with dying chickens allowing a sufficient source of infection for vertical transmission through horizontal transmission.

2.2 Susceptible animals When chicks are infected with this virus within 2 weeks of age, the rate of infection and morbidity is very high, and the eggs laid by infected hens are also highly virulent. If chicks aged 4–8 weeks are infected with this virus, the rate of morbidity and mortality is greatly reduced, and the eggs laid are not virulent. Chickens over 10 weeks of age do not have any morbidity after being infected, and the eggs laid are not virulent

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either.

2.3 Epidemic characteristics The disease occurs in chicken flocks all over the world, and the infection rate can be as high as 90% or more by the time of sexual maturity, but clinical morbidity and mortality are low.

3 Clinical Symptoms

Mental status: mental depression, cockscomb wattles pale, progressive emaciation (Fig.1–1, Fig.1–2, Fig.1–3).

Abdomen: abdomen enlarged.

Mouth: with tumor in mouth.

Eyes: eye discharge increased, aganoblepharon.

Skin: skin hair depilated, subcutaneous follicles bleeding(Fig.1–4).

4 Anatomical Symptoms

Abdominal cavity: blood clots visible in abdominal cavity after dissection of diseased chicken.

Heart: heart enlarged, with epicardial hemorrhage, myocardium with varying sized, grayish white to yellowish white tumors(Fig.2–1, Fig.2–2).

Liver: liver enlarged, with hemorrhagic necrosis and tumors(Fig.2–3, Fig.2–4).

Lungs: lungs with variable sized, grayish to yellowish white tumors .

Spleen: spleen enlarged with hemorrhagic necrosis and tumors (Fig.2–5, Fig.2–6).

Kidneys: kidneys with variable sized, grayish white to yellowish white tumors (Fig.2–7, Fig.2–8).

Adenogastric: Adenogastric erosion, nipple bleeding (Fig.2–9, Fig.2–10).

Intestines: intestines bleeding, with tumor lesions, mesentery with tumor nodules(Fig.2–11, Fig.2–12).

Reproductive organs: hens with tumors in ovaries (Fig.2–13), yolk hemorrhage; roosters with varying sizes of grayish–white to yellowish –white tumors in testis.

5 Diagnosis Methods

5.1 Clinical comprehensive diagnosis The disease usually occurs in chickens over 16 weeks of age, and the onset of the disease in chickens is progressive with consistently low mortality. Sick chickens clinically show almost no typical symptoms, with only progressive emaciation and pale cockscomb wattles. Some sick chickens have tumors in appearance, notably in the mouth and toes. Typical lesions on autopsy are enlarged and hemorrhagic internal organs with necrotic foci and grayish-white to yellowish-white tumors of varying sizes, most commonly in the liver, spleen, and reproductive organs, and laboratory diagnosis is required to confirm the diagnosis.

5.2 Laboratory diagnosis Laboratory diagnosis mainly includes virus isolation and cell culture, virus neutralization test, enzyme-linked immunosorbent assay, and molecular biology detection. Virus isolation and cell culture is simple but time-consuming, mainly used for the isolation of clinical materials; virus neutralization test has high specificity and can determine not only the genus but also the type, but the test must be carried out in the cell, which requires a more complex operating envi-

Fig.1 Clinical symptoms of avian leukosis

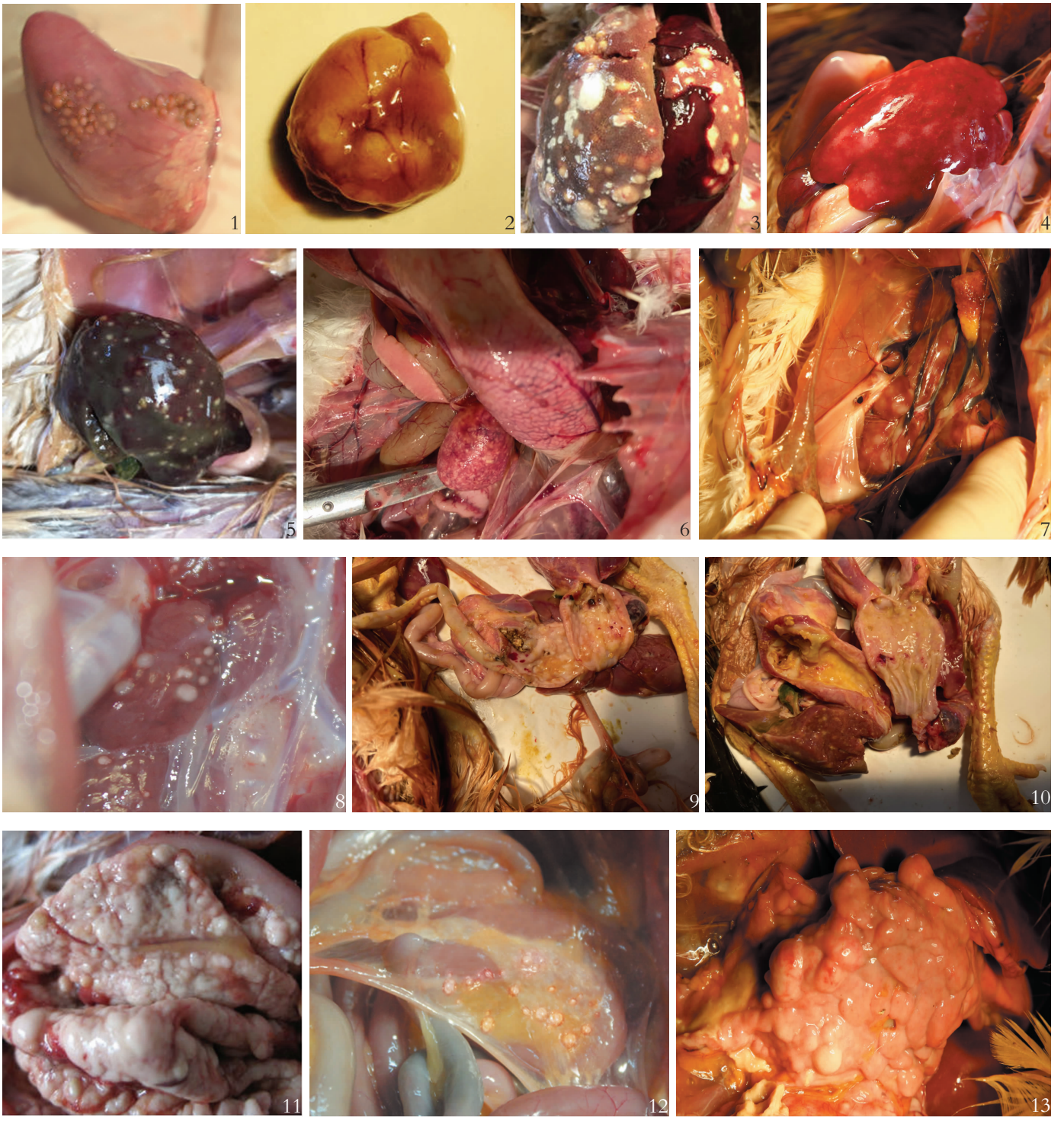

Fig.2 Pathological examination of avian leukosis

ronment and is time-consuming; due to simple operation, no requirement for special equipment and detection of a large number of samples in a short period of time, enzyme-linked immunosorbent assay can be used for epidemiological investigations and purification of chickens, but the test results are prone to false negatives or false positives; molecular biology diction is more commonly used in clinical practice because of high specificity and sensitivity, simplicity and convenience, and lower purity requirements for the test products.

6 Identification of Similar Diseases

6.1 Marek's disease of chicken Similarities: both diseases are contagious, and the affected chickens show clinical symptoms such as pale cockscomb wattles, loss of appetite, wasting, and emaciation; and

tumors of varying sizes can be seen in the internal organs by autopsy.

Differences: the pathogen of Marek's disease is Marek's disease virus, which mostly occurs in chickens between 8 and 12 weeks of age, while neurological, visceral, ophthalmological and cutaneous types can exist in the same flock; the tumors of diseased chickens are hard, and the bursa of Fabricius is atrophied or diffusely thickened (Fig.3).

6.2 Infectious bursal disease of chicken Similarities: both diseases are contagious, and the affected chickens show clinical symptoms such as mental instability, lethargy, loss of appetite, and dysentery; enlargement of the bursa of Fabricius is observed in autopsy in all cases.

Differences: the pathogen of infectious bursal disease is infectious bursal disease virus. Affected chickens show vent pecking, head and wings drooping, cold feeling, micro tremors and other clinical symptoms; dissection shows dark red hemorrhagic spots or blood spots on the pectoral and leg muscles (Fig.4–1, Fig.4–2), and the bursa of Fabricius is enlarged two to three times, round and hard, light yellow or obviously hemorrhagic, and purplish-red in severe cases, and the mucosa of the bursa of Fabricius has a jelly-like substance (Fig.4–3, Fig.4–4).

6.3 Reticuloendothelial hyperplasia of chicken Similarities: both diseases are contagious, and the affected chickens show clinical symptoms such as loss of appetite and depression; nodular hyperplasia of the spleen, liver, thymus, bursa of Fabricius, and pancreas are observed in all dissections.

Differences: the pathogen of chicken reticuloendothelial hyperplasia is reticulendotheliosus virus. Affected chickens show growth stagnation, abnormal feather growth, with small feather branches tightly attaching to the rhachis. Dissection reveals atrophy(Fig.5), congestion, and edema of the thymus; diffuse and nodular hyperplasia of r eticulocytes occurs in the liver, spleen, thymus, bursa of Fabricius, glandular stomach, and gonads. It can be determined by indirect fluorescent antibody test on 96-well culture plate.

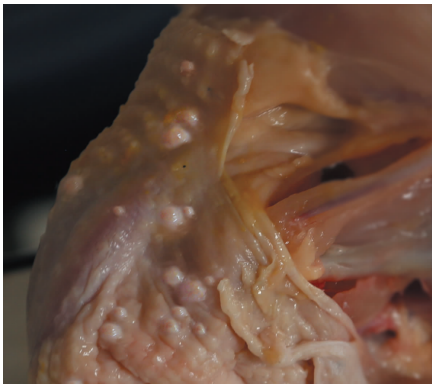

Fig.3 Pathological examination of Marek's disease of chicken

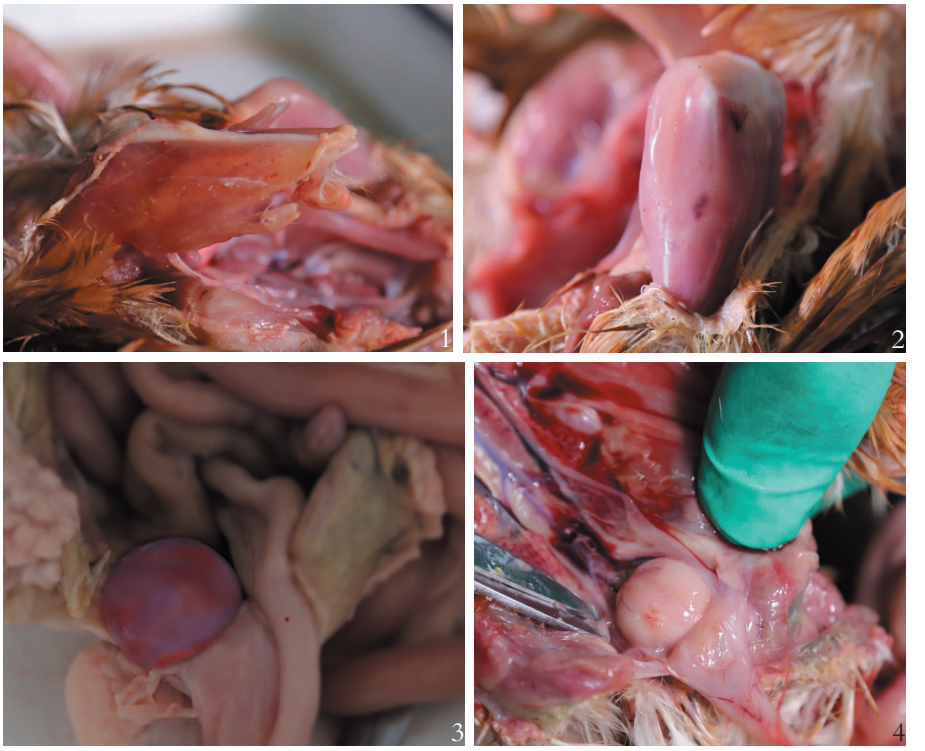

Fig.4 Pathological examination of infectious bursal disease of chicken

Fig.5 Pathological examination of reticuloendothelial hyperplasia of chicken

7 Prevention and Control Measures

7.1 Prevention measures

(1) Strengthe-ning feeding management. Sick chickens are fed with clean full-price feed and drinking water, and the chicken house is kept well ventilated at a suitable temperature; the management of incubation and brooding in the chicken house must be strengthened, especially closed and isolated rearing during the brooding period, and the system of "all in, all out" is implemented; the breeding eggs and embryos must be selected from the farms that do not have specific pathogens.

(2) Improving sanitation. The brooder house should be thoroughly fumigated and disinfected, and sterilized with chickens after entering the brooder house; hatching utensils should be thoroughly disinfected, and feces should be centrally handled to prevent the feed or drinking water from being contaminated; immunosuppressive diseases should be prevented by choosing and inoculating non-polluting vaccines at the appropriate time, to prevent the

chickens from being infected with avian leukosis virus as a result of the decline in their resistance.

(3) Strengthening quarantine and purification. The disease is mainly transmitted vertically. In the absence of an effective vaccine, we can only rely on sensitive diagnostic methods, and early diagnosis can eliminate virus-carrying eggs and breeders, so as to purify the breeding stock and reduce the transmission of the virus to the next generation through vertical transmission; enzyme-linked immunosorbent assay is performed on each batch of chickens about to lay eggs and positive chickens are eliminated; the disease will gradually disappear after 3 –4 generations of elimination.

7.2 Control measures Currently, there is no effective treatment for the disease. Reducing the infection rate in breeder flocks and establishing leukosis-free breeder flocks are the most effective measures to control the disease. Breeding hens are tested twice during the breeding period and twice during the egg-laying period, and positive hens should be eliminated. Eggs are collected for incubation from hens that are negative to avian leukosis, and then chicks are released and raised under isolation conditions for 4 consecutive generations to establish avian leukosis-purified flocks. However, due to long duration, high cost and complex technology, it is still difficult to be implemented in general breeding farms.

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